CUTEK Restore

Chemisys Manufacturing Pty Ltd

Chemwatch Hazard Alert Code: **4**

Version No: **1.1**
Safety Data Sheet according to WHS and ADG requirements

Issue Date: **16/12/2020**
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SECTION 1 Identification of the substance / mixture and of the company / undertaking

Product Identifier

Product name	CUTEK Restore
Chemical Name	Not Applicable
Synonyms	Not Available
Other means of identification	Not Available

Relevant identified uses of the substance or mixture and uses advised against

Relevant identified uses	Wood cleaner and restorer.

Details of the supplier of the safety data sheet

Registered company name	**Chemisys Manufacturing Pty Ltd**
Address	72 Chetwynd St QLD 4129 Australia
Telephone	+617 3188 5242
Fax	+617 3073 3919
Website	www.cutek.com.au
Email	admin@chemisys.com

Emergency telephone number

Association / Organisation	**Chemisys Manufacturing Pty Ltd**
Emergency telephone numbers	+617 3188 5246
Other emergency telephone numbers	131 126

SECTION 2 Hazards identification

Classification of the substance or mixture

HAZARDOUS CHEMICAL. NON-DANGEROUS GOODS. According to the WHS Regulations and the ADG Code.

ChemWatch Hazard Ratings

	Min	Max
Flammability	0	
Toxicity	0	
Body Contact	4	
Reactivity	0	
Chronic	3	

0 = Minimum
1 = Low
2 = Moderate
3 = High
4 = Extreme

Poisons Schedule	6
Classification [1]	Serious Eye Damage Category 1, Reproductive Toxicity Category 2, Skin Corrosion/Irritation Category 1A

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Legend:	*1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI*

Label elements

Hazard pictogram(s)	
Signal word	**Danger**

Hazard statement(s)

H361	Suspected of damaging fertility or the unborn child.
H314	Causes severe skin burns and eye damage.

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P260	Do not breathe mist/vapours/spray.
P280	Wear protective gloves/protective clothing/eye protection/face protection.
P281	Use personal protective equipment as required.

Precautionary statement(s) Response

P301+P330+P331	IF SWALLOWED: Rinse mouth. Do NOT induce vomiting.
P303+P361+P353	IF ON SKIN (or hair): Remove/Take off immediately all contaminated clothing. Rinse skin with water/shower.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P308+P313	IF exposed or concerned: Get medical advice/attention.
P310	Immediately call a POISON CENTER or doctor/physician.
P321	Specific treatment (see advice on this label).
P363	Wash contaminated clothing before reuse.
P304+P340	IF INHALED: Remove victim to fresh air and keep at rest in a position comfortable for breathing.

Precautionary statement(s) Storage

P405	Store locked up.

Precautionary statement(s) Disposal

P501	Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation.

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
144-62-7	<10	oxalic acid
111-76-2*	<10	2-Butoxyethanol
112-34-5	<10	diethylene glycol monobutyl ether
68081-81-2	<1	(C10-16)alkylbenzenesulfonic acid, sodium salt

SECTION 4 First aid measures

Description of first aid measures

Eye Contact	If this product comes in contact with the eyes:

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	‣ Immediately hold eyelids apart and flush the eye continuously with running water. ‣ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ‣ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. ‣ Transport to hospital or doctor without delay. ‣ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If skin or hair contact occurs: ‣ Immediately flush body and clothes with large amounts of water, using safety shower if available. ‣ Quickly remove all contaminated clothing, including footwear. ‣ Wash skin and hair with running water. Continue flushing with water until advised to stop by the Poisons Information Centre. ‣ Transport to hospital, or doctor.
Inhalation	‣ If fumes or combustion products are inhaled remove from contaminated area. ‣ Lay patient down. Keep warm and rested. ‣ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ‣ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ‣ Transport to hospital, or doctor, without delay.
Ingestion	‣ For advice, contact a Poisons Information Centre or a doctor at once. ‣ Urgent hospital treatment is likely to be needed. ‣ If swallowed do **NOT** induce vomiting. ‣ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. ‣ Observe the patient carefully. ‣ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. ‣ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. ‣ Transport to hospital or doctor without delay.

Indication of any immediate medical attention and special treatment needed

‣ Effective therapy against burns from oxalic acid involves replacement of calcium.
‣ Intravenous oxalic acid is substantially excreted (88% - 90%) in the urine within 36 hours.

For acute or short term repeated exposures to strong acids:

‣ Airway problems may arise from laryngeal edema and inhalation exposure. Treat with 100% oxygen initially.
‣ Respiratory distress may require cricothyroidotomy if endotracheal intubation is contraindicated by excessive swelling
‣ Intravenous lines should be established immediately in all cases where there is evidence of circulatory compromise.
‣ Strong acids produce a coagulation necrosis characterised by formation of a coagulum (eschar) as a result of the dessicating action of the acid on proteins in specific tissues.

INGESTION:

‣ Immediate dilution (milk or water) within 30 minutes post ingestion is recommended.
‣ **DO NOT** attempt to neutralise the acid since exothermic reaction may extend the corrosive injury.
‣ Be careful to avoid further vomit since re-exposure of the mucosa to the acid is harmful. Limit fluids to one or two glasses in an adult.
‣ Charcoal has no place in acid management.
‣ Some authors suggest the use of lavage within 1 hour of ingestion.

SKIN:

‣ Skin lesions require copious saline irrigation. Treat chemical burns as thermal burns with non-adherent gauze and wrapping.
‣ Deep second-degree burns may benefit from topical silver sulfadiazine.

EYE:

‣ Eye injuries require retraction of the eyelids to ensure thorough irrigation of the conjuctival cul-de-sacs. Irrigation should last at least 20-30 minutes. **DO NOT** use neutralising agents or any other additives. Several litres of saline are required.
‣ Cycloplegic drops, (1% cyclopentolate for short-term use or 5% homatropine for longer term use) antibiotic drops, vasoconstrictive agents or artificial tears may be indicated dependent on the severity of the injury.
‣ Steroid eye drops should only be administered with the approval of a consulting ophthalmologist).

[Ellenhorn and Barceloux: Medical Toxicology]

SECTION 5 Firefighting measures

Extinguishing media

‣ There is no restriction on the type of extinguisher which may be used.
‣ Use extinguishing media suitable for surrounding area.

Special hazards arising from the substrate or mixture

Fire Incompatibility	None known.

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Advice for firefighters

Fire Fighting	‣ Alert Fire Brigade and tell them location and nature of hazard. ‣ Wear breathing apparatus plus protective gloves in the event of a fire. ‣ Prevent, by any means available, spillage from entering drains or water courses. ‣ Use fire fighting procedures suitable for surrounding area. ‣ **DO NOT** approach containers suspected to be hot. ‣ Cool fire exposed containers with water spray from a protected location. ‣ If safe to do so, remove containers from path of fire. ‣ Equipment should be thoroughly decontaminated after use.
Fire/Explosion Hazard	‣ Non combustible. ‣ Not considered to be a significant fire risk. ‣ Acids may react with metals to produce hydrogen, a highly flammable and explosive gas. ‣ Heating may cause expansion or decomposition leading to violent rupture of containers. ‣ May emit corrosive, poisonous fumes. May emit acrid smoke. May emit poisonous fumes. May emit corrosive fumes.
HAZCHEM	Not Applicable

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	‣ Clean up all spills immediately. ‣ Avoid breathing vapours and contact with skin and eyes. ‣ Control personal contact with the substance, by using protective equipment. ‣ Contain and absorb spill with sand, earth, inert material or vermiculite. ‣ Wipe up. ‣ Place in a suitable, labelled container for waste disposal.
Major Spills	Chemical Class:acidic compounds, inorganic For release onto land: recommended sorbents listed in order of priority. SORBENT TYPE / RANK / APPLICATION / COLLECTION / LIMITATIONS LAND SPILL - SMALL foamed glass - pillows: 1 / throw / pitchfork / R, P, DGC, RT expanded mineral - particulate: 2 / shovel / shovel / R, I, W, P, DGC foamed glass - particulate: 2 / shovel / shovel / R, W, P, DGC LAND SPILL - MEDIUM expanded mineral -particulate: 1 / blower / skiploader / R, I, W, P, DGC foamed glass- particulate: 2 / blower / skiploader / R, W, P, DGC foamed glass - particulate: 3 / throw / skiploader / R, W, P, DGC Legend DGC: Not effective where ground cover is dense R; Not reusable I: Not incinerable P: Effectiveness reduced when rainy RT:Not effective where terrain is rugged SS: Not for use within environmentally sensitive sites W: Effectiveness reduced when windy Reference: Sorbents for Liquid Hazardous Substance Cleanup and Control; R.W Melvold et al: Pollution Technology Review No. 150: Noyes Data Corporation 1988 Moderate hazard. ‣ Clear area of personnel and move upwind. ‣ Alert Fire Brigade and tell them location and nature of hazard. ‣ Wear breathing apparatus plus protective gloves.

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	▸ Prevent, by any means available, spillage from entering drains or water course. ▸ Stop leak if safe to do so. ▸ Contain spill with sand, earth or vermiculite. ▸ Collect recoverable product into labelled containers for recycling. ▸ Neutralise/decontaminate residue (see Section 13 for specific agent). ▸ Collect solid residues and seal in labelled drums for disposal. ▸ Wash area and prevent runoff into drains. ▸ After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using. ▸ If contamination of drains or waterways occurs, advise emergency services.

Personal Protective Equipment advice is contained in Section 8 of the SDS.

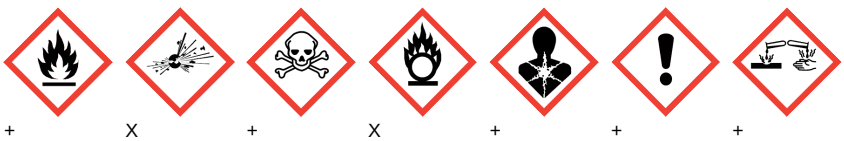
SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	▸ Avoid all personal contact, including inhalation. ▸ Wear protective clothing when risk of exposure occurs. ▸ Use in a well-ventilated area. ▸ Avoid contact with moisture. ▸ Avoid contact with incompatible materials. ▸ When handling, **DO NOT** eat, drink or smoke. ▸ Keep containers securely sealed when not in use. ▸ Avoid physical damage to containers. ▸ Always wash hands with soap and water after handling. ▸ Work clothes should be laundered separately. Launder contaminated clothing before re-use. ▸ Use good occupational work practice. ▸ Observe manufacturer's storage and handling recommendations contained within this SDS. ▸ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.
Other information	

Conditions for safe storage, including any incompatibilities

Suitable container	▸ Lined metal can, lined metal pail/ can. ▸ Plastic pail. ▸ Polyliner drum. ▸ Packing as recommended by manufacturer. ▸ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	Oxalic acid (and its dihydrate): ▸ react violently with strong oxidisers, bromine, furfuryl alcohol, hydrogen peroxide (90%), phosphorous trichloride, silver powders ▸ reacts explosively with chlorites and hypochlorites ▸ mixture with some silver compounds form explosive salts of silver oxalate ▸ is incompatible with caustics and alkalis, urea, alkaline metals and steel ▸ attacks polyvinyl alcohol and acetal plastics ▸ Avoid strong bases.



\+ X + X + + +

X — Must not be stored together
0 — May be stored together with specific preventions
+ — May be stored together

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure	oxalic acid	Oxalic acid	1 mg/m3	2 mg/m3	Not Available	Not Available

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Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Standards						
Australia Exposure Standards	2-Butoxyethanol	2-Butoxyethanol	20 ppm / 96.9 mg/m3	242 mg/m3 / 50 ppm	Not Available	Not Available

Emergency Limits

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
oxalic acid	Oxalic acid, anhydrous; (Ethanedioic acid)	2 mg/m3	20 mg/m3	500 mg/m3
2-Butoxyethanol	Butoxyethanol, 2-; (Glycol ether EB)	60 ppm	120 ppm	700 ppm
diethylene glycol monobutyl ether	Butoxyethoxy)ethanol, 2-(2-; (Diethylene glycol monobutyl ether)	30 ppm	33 ppm	200 ppm
(C10-16)alkylbenzenesulfonic acid, sodium salt	Sodium dodecylbenzenesulfonate; (Dodecyl benzene sodium sulfonate)	2.1 mg/m3	23 mg/m3	87 mg/m3

Ingredient	Original IDLH	Revised IDLH
oxalic acid	500 mg/m3	Not Available
2-Butoxyethanol	700 ppm	Not Available
diethylene glycol monobutyl ether	Not Available	Not Available
(C10-16)alkylbenzenesulfonic acid, sodium salt	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
diethylene glycol monobutyl ether	E	≤ 0.1 ppm
(C10-16)alkylbenzenesulfonic acid, sodium salt	E	≤ 0.01 mg/m³
Notes:	*Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.*	

MATERIAL DATA

There is only scant data regarding the toxicology of industrial exposure to airborne oxalates. There is no data regarding potential systemic toxicity or bioavailability of inhaled oxalates. The TLV-TWA (corresponding to 0.27 ppm on a molecular basis) is comparable to that of sulfuric acid and phosphoric acid and is thought to provide protection against the risk of eye and skin burns and respiratory tract irritation.
The recommendation for a STEL is added to prevent irritation of skin and mucous membranes.

For diethylene glycol monobutyl ether:
CEL TWA: 15.5 ppm, 100 mg/m3
(CEL = Chemwatch Exposure Limit)
In studies involving the inhalation toxicity of diethylene glycol monobutyl ether, exposure for 6 hours daily at 100 mg/m3 had no effect. This concentration is in the range of the saturated vapour concentration.
Local damage was produced following inhalation of concentrations higher than the saturated vapour concentrations, that is, during inhalation of the aerosol (350 mg/m3). Since the only potential effects of inhalation are restricted to local discomfort (in the aerosol concentration range) the substance is classified in category I for the limitation of exposure peaks.
Teratogenicity studies have not revealed prenatal toxic effects at high oral doses and this ether is classified in pregnancy risk group C.

Exposure controls

Appropriate engineering controls	Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection. The basic types of engineering controls are: Process controls which involve changing the way a job activity or process is done to reduce the risk. Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use. Employers may need to use multiple types of controls to prevent employee overexposure. Local exhaust ventilation usually required. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection. Supplied-air type respirator may be required in special circumstances. Correct fit is essential to ensure adequate protection. An approved self contained breathing apparatus (SCBA) may be required in some situations. Provide adequate ventilation in warehouse or closed storage area. Air contaminants generated in the workplace possess varying

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	"escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant. Type of Contaminant: / Air Speed: solvent, vapours, degreasing etc., evaporating from tank (in still air). / 0.25-0.5 m/s (50-100 f/min.) aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation) / 0.5-1 m/s (100-200 f/min.) direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion) / 1-2.5 m/s (200-500 f/min.) grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion). / 2.5-10 m/s (500-2000 f/min.) Within each range the appropriate value depends on: Lower end of the range / Upper end of the range 1: Room air currents minimal or favourable to capture / 1: Disturbing room air currents 2: Contaminants of low toxicity or of nuisance value only. / 2: Contaminants of high toxicity 3: Intermittent, low production. / 3: High production, heavy use 4: Large hood or large air mass in motion / 4: Small hood-local control only Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.
Personal protection	
Eye and face protection	‣ Safety glasses with unperforated side shields may be used where continuous eye protection is desirable, as in laboratories; spectacles are not sufficient where complete eye protection is needed such as when handling bulk-quantities, where there is a danger of splashing, or if the material may be under pressure. ‣ Chemical goggles.whenever there is a danger of the material coming in contact with the eyes; goggles must be properly fitted. ‣ Full face shield (20 cm, 8 in minimum) may be required for supplementary but never for primary protection of eyes; these afford face protection. ‣ Alternatively a gas mask may replace splash goggles and face shields. ‣ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]
Skin protection	See Hand protection below
Hands/feet protection	‣ Elbow length PVC gloves ‣ When handling corrosive liquids, wear trousers or overalls outside of boots, to avoid spills entering boots.
Body protection	See Other protection below
Other protection	‣ Overalls. ‣ P.V.C apron. ‣ Barrier cream. ‣ Skin cleansing cream. ‣ Eye wash unit.

Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:
"Forsberg Clothing Performance Index".
The effect(s) of the following substance(s) are taken into account in the ***computer-generated*** selection:
CUTEK Restore

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Material	CPI
BUTYL	A
NEOPRENE	A
VITON	A
NATURAL RUBBER	C
NATURAL+NEOPRENE	C
NEOPRENE/NATURAL	C
NITRILE	C
NITRILE+PVC	C
PVA	C
PVC	C

* CPI - Chemwatch Performance Index
A: Best Selection
B: Satisfactory; may degrade after 4 hours continuous immersion
C: Poor to Dangerous Choice for other than short term immersion
NOTE: As a series of factors will influence the actual performance of the glove, a final selection must be based on detailed observation. -
* Where the glove is to be used on a short term, casual or infrequent basis, factors such as "feel" or convenience (e.g. disposability), may dictate a choice of gloves which might otherwise be unsuitable following long-term or frequent use. A qualified practitioner should be consulted.

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Not Available

Physical state	Liquid	**Relative density (Water = 1)**	1.09
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Available
pH (as supplied)	1.5	**Decomposition temperature**	Not Available
Melting point / freezing point (°C)	Not Available	**Viscosity (cSt)**	Not Available
Initial boiling point and boiling range (°C)	Not Available	**Molecular weight (g/mol)**	Not Available
Flash point (°C)	Not Available	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	Not Available	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	Not Available	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	Not Available	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	Not Available	**Gas group**	Not Available
Solubility in water	Miscible	**pH as a solution (1%)**	Not Available
Vapour density (Air = 1)	Not Available	**VOC g/L**	Not Available

SECTION 10 Stability and reactivity

Reactivity	See section 7
Chemical stability	‣ Unstable in the presence of incompatible materials. ‣ Product is considered stable. ‣ Hazardous polymerisation will not occur.
Possibility of hazardous reactions	See section 7

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Conditions to avoid	See section 7
Incompatible materials	See section 7
Hazardous decomposition products	See section 5

SECTION 11 Toxicological information

Information on toxicological effects

Inhaled	Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system. Acidic corrosives produce respiratory tract irritation with coughing, choking and mucous membrane damage. Symptoms of exposure may include dizziness, headache, nausea and weakness. In more severe exposures, pulmonary oedema may be evident either immediately or after a latent period of 5-72 hours. Symptoms of pulmonary oedema include a tightness in the chest, dyspnoea, frothy sputum and cyanosis. Examination may reveal hypotension, a weak and rapid pulse and moist rates. Death, due to anoxia, may occur several hours after onset of the pulmonary oedema. Inhalation of oxalic acid dusts or vapours can cause ulceration of the mucous membranes of the nose and throat, epistaxis (nosebleed), headache and nervousness. The airborne dust behaves as a strong acid producing severe local burns of the mucous membranes. The material has **NOT** been classified by EC Directives or other classification systems as "harmful by inhalation". This is because of the lack of corroborating animal or human evidence. In the absence of such evidence, care should be taken nevertheless to ensure exposure is kept to a minimum and that suitable control measures be used, in an occupational setting to control vapours, fumes and aerosols.
Ingestion	Ingestion of acidic corrosives may produce circumoral burns with a distinct discolouration of the mucous membranes of the mouth, throat and oesophagus. Immediate pain and difficulties in swallowing and speaking may also be evident. Oedema of the epiglottis may produce respiratory distress and possibly, asphyxia. Nausea, vomiting, diarrhoea and a pronounced thirst may occur. More severe exposures may produce a vomitus containing fresh or dark blood and large shreds of mucosa. Shock, with marked hypotension, weak and rapid pulse, shallow respiration and clammy skin may be symptomatic of the exposure. Circulatory collapse may, if left untreated, result in renal failure. Severe cases may show gastric and oesophageal perforation with peritonitis, fever and abdominal rigidity. Stricture of the oesophageal, gastric and pyloric sphincter may occur as within several weeks or may be delayed for years. Death may be rapid and often results from asphyxia, circulatory collapse or aspiration of even minute amounts. Delayed deaths may be due to peritonitis, severe nephritis or pneumonia. Coma and convulsions may be terminal. Oxalic acid is a minor, normal body constituent occurring in blood at approximately 0.150 mg/100 ml and in kidney, muscle and liver at about 0.050 mg/100 ml dry weight, but higher concentrations are toxic. Ingestion of 5 grams has caused death within hours. It is a systemic poison which affects the central nervous system and kidney function. Low doses (i.e. excess in blood) may cause hypocalcemia (presence in the blood of an abnormally low concentration of calcium). Oxalic acid occurs naturally in the common weed Oxalis, `sour sobs'. The material has **NOT** been classified by EC Directives or other classification systems as "harmful by ingestion". This is because of the lack of corroborating animal or human evidence. The material may still be damaging to the health of the individual, following ingestion, especially where pre-existing organ (e.g liver, kidney) damage is evident. Present definitions of harmful or toxic substances are generally based on doses producing mortality rather than those producing morbidity (disease, ill-health). Gastrointestinal tract discomfort may produce nausea and vomiting. In an occupational setting however, ingestion of insignificant quantities is not thought to be cause for concern.
Skin Contact	Skin contact with acidic corrosives may result in pain and burns; these may be deep with distinct edges and may heal slowly with the formation of scar tissue. Skin contact is not thought to have harmful health effects (as classified under EC Directives); the material may still produce health damage following entry through wounds, lesions or abrasions. Solutions of 5% to 10% oxalic acid are irritating to the skin after prolonged contact; early gangrene may occur after hand immersion in oxalate solutions. Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.
Eye	Direct eye contact with acid corrosives may produce pain, lachrymation, photophobia and burns. Mild burns of the epithelia generally recover rapidly and completely. Severe burns produce long-lasting and possible irreversible damage. The appearance of the burn may not be apparent for several weeks after the initial contact. The cornea may ultimately become deeply vascularised and opaque resulting in blindness. When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation. Irritation of the eyes may produce a heavy secretion of tears (lachrymation).

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Chronic	Repeated or prolonged exposure to acids may result in the erosion of teeth, inflammatory and ulcerative changes in the mouth and necrosis (rarely) of the jaw. Bronchial irritation, with cough, and frequent attacks of bronchial pneumonia may ensue. Gastrointestinal disturbances may also occur. Chronic exposures may result in dermatitis and/or conjunctivitis. The impact of inhaled acidic agents on the respiratory tract depends upon a number of interrelated factors. These include physicochemical characteristics, e.g., gas versus aerosol; particle size (small particles can penetrate deeper into the lung); water solubility (more soluble agents are more likely to be removed in the nose and mouth). Given the general lack of information on the particle size of aerosols involved in occupational exposures to acids, it is difficult to identify their principal deposition site within the respiratory tract. Acid mists containing particIes with a diameter of up to a few micrometers will be deposited in both the upper and lower airways. They are irritating to mucous epithelia, they cause dental erosion, and they produce acute effects in the lungs (symptoms and changes in pulmonary function). AsthmatIcs appear to be at particular risk for pulmonary effects. Repeated or long-term occupational exposure is likely to produce cumulative health effects involving organs or biochemical systems. Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems. Exposure to the material may cause concerns for humans owing to possible developmental toxic effects, generally on the basis that results in appropriate animal studies provide strong suspicion of developmental toxicity in the absence of signs of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not a secondary non-specific consequence of other toxic effects.

CUTEK Restore	**TOXICITY**	**IRRITATION**
	Not Available	Not Available

oxalic acid	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 2000 mg/kg[2]	Eye: adverse effect observed (irritating)[1]
	Oral(Rat) LD50 ~660-700 mg/kg[2]	Skin: no adverse effect observed (not irritating)[1]
	Oral(Rat) LD50 =375 mg/kg[2]	
	Oral(Rat) LD50 475 mg/kg[2]	

2-Butoxyethanol	**TOXICITY**	**IRRITATION**
	90-1800 mg/kg[2]	Eye: adverse effect observed (irritating)[1]
	Inhalation(Rat) LC50 2.21 mg/l**[2]	Skin: adverse effect observed (irritating)[1]
	Inhalation(Rat) LC50 449.48655 mg/l/4H[2]	Skin: no adverse effect observed (not irritating)[1]
	Oral(Guinea) LD50 =1200 mg/kg[2]	
	Oral(Mouse) LD50 =1000-1600 mg/kg[2]	
	Oral(Mouse) LD50 =1230 mg/kg[2]	
	Oral(Mouse) LD50 =1700 mg/kg[2]	
	Oral(Rabbit) LD50 =320 mg/kg[2]	
	Oral(Rat) LD50 =1190-2800 mg/kg[2]	
	Oral(Rat) LD50 =1480 mg/kg[2]	
	Oral(Rat) LD50 =470 mg/kg[2]	
	Oral(Rat) LD50 250 mg/kg[2]	

diethylene glycol monobutyl ether	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 4120 mg/kg[2]	Eye (rabbit): 20 mg/24h moderate
	Oral(Guinea) LD50 =1720-2310 mg/kg[2]	Eye (rabbit): 5 mg - SEVERE
	Oral(Mouse) LD50 =5526 mg/kg[2]	
	Oral(Rabbit) LD50 =2200 mg/kg[2]	
	Oral(Rat) LD50 =4500 mg/kg[2]	
	Oral(Rat) LD50 =5080 mg/kg[2]	
	Oral(Rat) LD50 =5660 mg/kg[2]	

(C10-16)alkylbenzenesulfonic acid, sodium salt	**TOXICITY**	**IRRITATION**
	Oral(Rat) LD50 438 mg/kg[2]	Eye (rabbit): 0.25 mg/24hr-SEVERE
		Eye (rabbit): 1% - SEVERE
		Eye: adverse effect observed (irritating)[1]

Continued...

		Skin (rabbit): 20 mg/24 hr-SEVERE
		Skin: adverse effect observed (corrosive)[1]
		Skin: no adverse effect observed (not irritating)[1]

Legend:	*1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2.* Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances*

CUTEK Restore	for acid mists, aerosols, vapours Data from assays for genotoxic activity in vitro suggest that eukaryotic cells are susceptible to genetic damage when the pH falls to about 6.5. Cells from the respiratory tract have not been examined in this respect. Mucous secretion may protect the cells of the airways from direct exposure to inhaled acidic mists, just as mucous plays an important role in protecting the gastric epithelium from its auto-secreted hydrochloric acid. In considering whether pH itself induces genotoxic events in vivo in the respiratory system, comparison should be made with the human stomach, in which gastric juice may be at pH 1-2 under fasting or nocturnal conditions, and with the human urinary bladder, in which the pH of urine can range from <5 to > 7 and normally averages 6.2. Furthermore, exposures to low pH in vivo differ from exposures *in vitro* in that, *in vivo*, only a portion of the cell surface is subjected to the adverse conditions, so that perturbation of intracellular homeostasis may be maintained more readily than in vitro.
DIETHYLENE GLYCOL MONOBUTYL ETHER	The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. For diethylene glycol monoalkyl ethers and their acetates: This category includes diethylene glycol ethyl ether (DGEE), diethylene glycol propyl ether (DGPE) diethylene glycol butyl ether (DGBE) and diethylene glycol hexyl ether (DGHE) and their acetates. **Acute toxicity:** There are adequate oral, inhalation and/or dermal toxicity studies on the category members. Oral LD50 values in rats for all category members are all > 3000 mg/kg bw, with values generally decreasing with increasing molecular weight. Four to eight hour acute inhalation toxicity studies were conducted for all category members except DGPE in rats at the highest vapour concentrations achievable. No lethality was observed for any of these materials under these conditions. Dermal LD50 values in rabbits range from 2000 mg/kg bw (DGHE) to 15000 mg/kg bw (DGEEA). Signs of acute toxicity in rodents are consistent with non-specific CNS depression typical of organic solvents in general. All category members are slightly irritating to skin and slightly to moderately irritating to eyes (with the exception of DGHE, which is highly irritating to eyes). Sensitisation tests with DGEE, DGEEA, DGPE, DGBE and DGBEA in animals and/or humans were negative. **Repeat dose toxicity**: Valid oral studies conducted using DGEE, DGPE, DGBEA, DGHE and the supporting chemical DGBE ranged in duration from 30 days to 2 years. Effects predominantly included kidney and liver toxicity, absolute and/or relative changes in organ weights, and some changes in haematological parameters. All effects were seen at doses greater than 800-1000 mg/kg bw/day from oral or dermal studies; no systemic effects were observed in inhalation studies with less than continuous exposure regimens. **Mutagenicity:** DGEE, DGEEA, DGBE, DGBEA and DGHE generally tested negative for mutagenicity in *S. typhimurium* strains TA98, TA100, TA1535, TA1537 and TA1538 and DGBEA tested negative in E. coli WP2uvrA, with and without metabolic activation. *In vitro* cytogenicity and sister chromatid exchange assays with DGBE and DGHE in Chinese Hamster Ovary Cells with and without metabolic activation and *in vivo* micronucleus or cytogenicity tests with DGEE, DGBE and DGHE in rats and mice were negative, indicating that these diethylene glycol ethers are not likely to be genotoxic. **Reproductive and developmental toxicity:** Reliable reproductive toxicity studies on DGEE, DGBE and DGHE show no effect on fertility at the highest oral doses tested (4,400 mg/kg/day for DGEE in the mouse and 1,000 mg/kg/day for DGBE and DGHE in the rat). The dermal NOAEL for reproductive toxicity in rats administered DGBE also was the highest dose tested (2,000 mg/kg/day). Although decreased sperm motility was noted in F1 mice treated with 4,400 mg/kg/day DGEE in drinking water for 14 weeks, sperm concentrations and morphology, histopathology of the testes and fertility were not affected. Results of the majority of adequate repeated dose toxicity studies in which reproductive organs were examined indicate that DGPE and DGBEA do not cause toxicity to reproductive organs (including the testes). Test material-related testicular toxicity was not noted in the majority of the studies with DGEE or DGEEA. Results of the developmental toxicity studies conducted with DGEE, DGBE and DGHE are almost exclusively negative. In these studies, effects on the foetus are generally not observed (even at concentrations that produced maternal toxicity). Exposure to 102 ppm (560 mg/m3) DGEE by inhalation (maximal achievable vapour concentration) or 1385 mg/kg/day DGEE by the dermal route during gestation did not cause maternal or developmental toxicity in the rat. Maternal toxicity and teratogenesis were not observed in rabbits receiving up to 1000 mg/kg/day DGBE by the dermal route during gestation; however a transient decrease in body weight was observed, which reversed by Day 21 In the mouse, the only concentration of DGEE tested (3500 mg/kg/day by gavage) caused maternal, but no foetal toxicity. Also, whereas oral administration of 2050 mg/kg/day DGBE (gavage) to the mouse and 1000 mg/kg/day DGHE (dietary) caused maternal toxicity, these doses had no effect on the developing foetus
(C10-16)ALKYLBENZENESULFONIC ACID, SODIUM SALT	for alkaryl sulfonate petroleum additives: **Mammalian Toxicology - Acute.** Existing data on acute mammalian toxicity indicates a low concern for acute toxicity. Acute oral toxicity: In all but one studies, there were no deaths that could be attributed to treatment with the test material when administered at the limit dose of 2000 or 5000 mg/kg. In some studies, the primary clinical observations were diarrhea and reduced food consumption (without a change in body weight). These effects are consistent with the gastrointestinal irritant properties of detergents in an oil-based vehicle. In other studies, decreased body weight gain or ruffled fur was observed. In one study where deaths occurred, animals were administered dose levels well above the 2000 mg/kg limit dose. Overall, the acute oral LD50 for these substances was greater than the 2000 mg/kg limit dose indicating a relatively low order of toxicity.

Continued...

Acute dermal toxicity: No mortality was observed for any tested substance when administered at the limit dose of 2000 or 5000 mg/kg. The principal clinical observation was erythema and/or edema at the site of dermal application. In some cases, the cutaneous findings included dry, flaky skin, desquamation and hyperkeratosis. Overall, the acute dermal LD50 for these substances was greater than the 2000 mg/kg limit dose indicating a relatively low order of toxicity.
Acute inhalation toxicity: One member of the petroleum additive alkaryl sulfonate category (CAS RN: 6878396-0) was tested for acute inhalation toxicity (OECD Guideline 403, Acute Inhalation Toxicity). Rats were exposed whole-body to an aerosol of the substance at a nominal atmospheric concentration of 1.9 mg/L for four hours. This was the maximum attainable concentration due to the low volatility and high viscosity of the test material. No mortality was noted, and all animals fully recovered following depuration. Clinical signs of toxicity during exposure included reduced activity, matted coat, and closed eyes. Clinical signs of toxicity observed post exposure included lacrimation, nasal discharge, salivation rates, matted coat, hunched appearance, soft stools and closed eyes. No treatment-related macroscopic findings were noted. The lack of mortality at a concentration just below the limit dose of 2.0 mg/L indicates a relatively low order of toxicity for this substance.
Mammalian Toxicology - Subchronic Toxicity. Existing data from repeated-dose toxicity studies indicates minimal signs of toxicity following repeated oral exposure. Adverse effects at the site of contact were observed following repeated dermal exposure (injury to the skin) and repeated inhalation (injury to the lungs).
NOAELs rage from 49.5 mg/m3 to 1000 mg/kg/day
Mammalian Toxicology - Reproductive and Developmental Toxicity. A one-generation reproductive toxicity test was conducted on one member of the category (CAS # 115733-09-0). Exposure to the alkaryl sulfonate did not significantly impact reproduction or development and these results were bridged to the remainder of the category.
Mammalian Toxicology - Mutagenicity. Existing data from bacterial reverse mutation assays and in vitro and in vivo chromosome aberration studies indicate a low concern for mutagenicity.
Animal Irritation
An acute eye irritation study indicates that calcium dodecylbenzenesulfonate caused irritation.
Result: irritating at 0.1 ml
An acute skin study indicate that calcium dodecylbenzenesulfonate is irritant to skin 0.5 ml according to OECD GHS guidelines.
Respiratory irritation was not observed. There were no treatment-related changes in the haematological or urinalysis values in any of the animals. No signs of irritation of respiratory tract and nasal effects were observed.
The material may produce severe skin irritation after prolonged or repeated exposure, and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) thickening of the epidermis.
Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. Prolonged contact is unlikely, given the severity of response, but repeated exposures may produce severe ulceration.
Linear alkylbenzene sulfonates (LAS) are classified as Irritant (Xi) with the risk phrases R38 (Irritating to skin) and R41 (Risk of serious damage to eyes) according to CESIO (CESIO 2000). LAS are not included in Annex 1 of list of dangerous substances of Council Directive 67/548/EEC.
Linear alkylbenzene sulfonic acids (LABS) are strong acids (pKa<2) are classified as corrosive (R34)
Branched materials exhibit comparable toxicity to linear species.
Acute toxicity: The available data indicate minimal to moderate toxicity, with LD50 values ranging from 500 to 2000 mg/kg body weight (bw). Acute inhalation data also indicate a lack of significant toxicity.Available dermal exposure data also shows a lack of significant toxicity.
LAS are readily absorbed by the gastrointestinal tract after oral administration in animals. LAS are not readily absorbed through the skin . The bulk is metabolised in the liver to sulfophenylic carboxyl acids. The metabolites are excreted primarily via the urine and faeces. The main urinary metabolites in rats are sulfophenyl butanoic acid and sulfophenyl pentanoic acid. Accumulation of LAS or its main metabolites has not been established in any organ after repeated oral ingestion.
No serious injuries or fatalities in man have been reported following accidental ingestion of LAS-containing detergent. The main clinical signs observed after oral administration to rats of doses near or greater than the LD50 values consisted of reduced voluntary activity, diarrhoea, weakness etc. Death usually occurred within 24 hours of administration. Rats appear to be more sensitive to LAS than mice.
LAS and branched alkylbenzene sulfonates may cause irritation of the eyes, skin and mucous membranes. LAS are relatively more irritating to the skin than the corresponding branched alkylbenzene sulfonates. The potential of LAS to irritate the skin depends on the concentration applied. LAS have been classified as irritating to skin at concentrations above 20% according to EU-criteria. Human skin can tolerate contact with solution of up to 1% LAS for 24 hours resulting in only mild irritation. Application of > 5% LAS to the eyes of rabbits produced irritation. Concentration of < 0.1% LAS produced mild to no irritation.
Skin sensitization was not seen in 2,294 volunteers exposed to LAS or in 17,887 exposed to formulations of LAS.
Repeat dose toxicity: A feeding study indicated that LAS, when administered for 2 years at extremely high levels (0.5%) in the diets to rats, produced no adverse effects on growth, health or feed efficiency.
Genotoxicity: The mutagenic potential of LAS was tested using *Salmonella typhimurium* strains, using Ames test. In these studies, LAS was not mutagenic. The available long-term studies are inadequate for evaluating the carcinogenic potential of LAS in laboratory animals. The studies available (oral administration to rats and mice) do not show any evidence of carcinogenicity.
Reproductive toxicity: In general no specific effect of LAS on reproductive processes has been seen, although dosages causing maternal toxicity may also induce some effects on reproduction. No teratogenic effects attributed to LAS exposure have been observed.
Environmental and Health Assessment of Substances in Household Detergents and Cosmetic Detergent Products, Environment Project, 615, 2001. Torben Madsen et al: Miljoministeriet (Danish Environmental Protection Agency)
For aromatic sulfonic acids
Aromatic sulfonic acids are very corrosive as was demonstrated in skin and eye irritation studies, in the acute oral

Continued...

	studies, and in the single repeated dose oral study. Health records from industrial manufacturing exposure, including manufacturing plant book of injuries and a physician report, show toluene-4-sulphonic acid (as handled in manufacturing plants; i.e., a 65% aqueous solution with < 5% free sulphuric acid) is an irritant to the eye and skin. **Sensitisation:** There is a single, key study for sensitization of the aromatic sulphonic acids. None of the tested animals showed positive responses in a, well documented, GLP guinea pig sensitization study with toluene-4-sulphonic acid (CAS No. 104-15-4). The test substance can be considered a non-sensitizer in guinea pigs as none of the test animals showed a positive response to combined intradermal and topical induction followed by topical challenge. **Repeat dose toxicity:** A GLP guideline study with p-toluenesulphonic acid (CAS No. 104-15-4) reported no adverse effects to male and female rats exposed orally for 28 days. The highest dose was 500 mg/kg bw/day (>490 mg/kg bw/day based on >98% active ingredient). Therefore the NOAEL was set at 500 mg/kg bw/day. Toxicity to reproduction: No fertility studies are reported for the aromatic sulphonic acids. There are however studies for the chemically related hydrotrope substances that looked at reproductive organs and development of offspring. Hydrotropes are the salt form of the sulphonic acids and therefore are used as read-across for this endpoint. The 90-day oral rat and oral mouse studies and the 2-year chronic dermal rat and mouse studies with the closely related compound sodium xylene sulfonate (CAS No. 1300-72-7) included examination of sex organs of both sexes. No treatment related effects on reproductive organs were reported at doses roughly equivalent to those in the developmental toxicity study. he NOAEL for both maternal and foetal toxicity was the highest dose tested - 3000 mg/kg bw /day which is equivalent to 936 mg active ingredient per kilogram body weight per day. The conclusion of the study was no indications of developmental toxicity including teratogenesis. **Genetic toxicity:** There is a fully documented, GLP Guideline (OECD 471) Ames Test and a fully documented, GLP Guideline (OECD 473) Chromosome Aberration Test for one of the aromatic sulphonic acids, p-toluenesulphonic acid (CAS No. 104-15-4). Both tests were conducted with and without metabolic activation. The Ames test exposed up to 5000 micrograms/plate and the chromosome aberration test exposed up to 1902 micrograms per liter of the test substance. These studies conclude the substance is neither mutagenic norcytotoxic. There is an additional, published report of an Ames Test for another of the aromatic sulphonic acids, benzenesulfonic acid (CAS No. 98-11-3). Exposures up to 10,000 micrograms/plate were done with and without metabolic activation. The conclusion is the same as for the p-toluenesulphonic acid; that is, not mutagenic and not cytotoxic. There are no in vivo mutagenicity studies for the aromatic sulphonic acids, but there are two in vivo mouse micronucleus studies for the related hydrotropes – sodium cumene sulfonate (CAS 28348-53-0) and calcium xylene sulfonate (CAS 28088-63-3). Both are GLP-compliant Guideline mouse micronucleus studies with full documentation. Both studies conclude the test substances were not mutagenic in these assays. Disulfonic acids have not been the subject of concern. **Carcinogenicity:** There are no carcinogenicity studies for the aromatic sulphonic acids Two hydrotrope studies involve 2-year rat and mouse dermal exposures conducted under GLP. Up to 240 mg (rats) and 727 mg (mice) sodium xylenesulfonate/kg body weight in 50% ethanol were dosed 5 days per week for 104 weeks. There were no treatment related incidences of mononuclear cell leukenia, neoplasms, or nonneoplatic lesions of the skin and other organs. The increased incidence of epidermal hyperplasia may have been related to exposure to the test substance. The NOAEL was reported as 240 mg/kg bw/day for rats and 727 mg/kg bw/day for mice. **Elimination:** The US EPA has evaluated the metabolism of analogs in in the sodium alkyl naphthalenesulfonate cluster (SANS), a group of sodium salts of naphthalenesulfonic acids . In a US EPA final rule for SANS, it was stated that “the 1- or 2-sulfonic acid sodium salt moieties on the naphthalene ring may provide a handle by which these compounds can be readily conjugated and eliminated.”
CUTEK Restore & OXALIC ACID & (C10-16)ALKYLBENZENESULFONIC ACID, SODIUM SALT	Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.

Acute Toxicity	✗	**Carcinogenicity**	✗
Skin Irritation/Corrosion	✓	**Reproductivity**	✓
Serious Eye Damage/Irritation	✓	**STOT - Single Exposure**	✗
Respiratory or Skin sensitisation	✗	**STOT - Repeated Exposure**	✗
Mutagenicity	✗	**Aspiration Hazard**	✗

Continued...

Legend: ✗ *– Data either not available or does not fill the criteria for classification*
✓ *– Data available to make classification*

SECTION 12 Ecological information

Toxicity

CUTEK Restore	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	Not Available	Not Available	Not Available	Not Available	Not Available
oxalic acid	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50	48	Crustacea	-125-150mg/L	4
	EC50	72	Algae or other aquatic plants	>18.39-<19.92mg/L	2
	NOEC	0.33	Algae or other aquatic plants	-0.002-0.003emol/dm3	4
2-Butoxyethanol	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	LC50	96	Fish	1250-mg/L	4
	EC50	48	Crustacea	164mg/L	2
	EC50	72	Algae or other aquatic plants	623mg/L	2
	NOEL	336	Not Available	49.50000-mg/L	4
diethylene glycol monobutyl ether	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	LC50	96	Fish	1300-mg/L	4
	EC50	48	Crustacea	>100mg/L	2
	EC50	96	Algae or other aquatic plants	>100mg/L	2
	NOEC	96	Algae or other aquatic plants	>=100mg/L	1
(C10-16)alkylbenzenesulfonic acid, sodium salt	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	NOEC	72	Crustacea	1mg/L	5
Legend:	*Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. EPIWIN Suite V3.12 (QSAR) - Aquatic Toxicity Data (Estimated) 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data*				

Ecotoxicity:
The tolerance of water organisms towards pH margin and variation is diverse. Recommended pH values for test species listed in OECD guidelines are between 6.0 and almost 9. Acute testing with fish showed 96h-LC50 at about pH 3.5
DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
oxalic acid	LOW	LOW
2-Butoxyethanol	LOW (Half-life = 56 days)	LOW (Half-life = 1.37 days)
diethylene glycol monobutyl ether	LOW	LOW

Bioaccumulative potential

Ingredient	Bioaccumulation
oxalic acid	LOW (LogKOW = -1.7365)
2-Butoxyethanol	LOW (BCF = 2.51)
diethylene glycol monobutyl ether	LOW (BCF = 0.46)

Mobility in soil

Continued...

Ingredient	Mobility
oxalic acid	HIGH (KOC = 1.895)
2-Butoxyethanol	HIGH (KOC = 1)
diethylene glycol monobutyl ether	LOW (KOC = 10)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	▸ Recycle wherever possible. ▸ Consult manufacturer for recycling options or consult local or regional waste management authority for disposal if no suitable treatment or disposal facility can be identified. ▸ Treat and neutralise at an approved treatment plant. Treatment should involve: Neutralisation with soda-ash or soda-lime followed by: burial in a land-fill specifically licensed to accept chemical and / or pharmaceutical wastes or Incineration in a licensed apparatus (after admixture with suitable combustible material). ▸ Decontaminate empty containers with 5% aqueous sodium hydroxide or soda ash, followed by water. Observe all label safeguards until containers are cleaned and destroyed.

SECTION 14 Transport information

Labels Required

Marine Pollutant	NO
HAZCHEM	Not Applicable

Land transport (ADG): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

Air transport (ICAO-IATA / DGR): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

Sea transport (IMDG-Code / GGVSee): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

Transport in bulk according to Annex II of MARPOL and the IBC code

Not Applicable

SECTION 15 Regulatory information

Safety, health and environmental regulations / legislation specific for the substance or mixture

oxalic acid is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
Australian Inventory of Industrial Chemicals (AIIC)

2-Butoxyethanol is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
Australian Inventory of Industrial Chemicals (AIIC)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

diethylene glycol monobutyl ether is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Australian Inventory of Industrial Chemicals (AIIC)

(C10-16)alkylbenzenesulfonic acid, sodium salt is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Australian Inventory of Industrial Chemicals (AIIC)

National Inventory Status

Continued...

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (oxalic acid; 2-Butoxyethanol; diethylene glycol monobutyl ether; (C10-16)alkylbenzenesulfonic acid, sodium salt)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	Yes
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	Yes
Vietnam - NCI	Yes
Russia - ARIPS	Yes
Legend:	*Yes = All CAS declared ingredients are on the inventory* *No = One or more of the CAS listed ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)*

SECTION 16 Other information

Revision Date	16/12/2020
Initial Date	07/12/2020

Other information

Classification of the preparation and its individual components has drawn on official and authoritative sources as well as independent review by the Chemwatch Classification committee using available literature references.
The SDS is a Hazard Communication tool and should be used to assist in the Risk Assessment. Many factors determine whether the reported Hazards are Risks in the workplace or other settings. Risks may be determined by reference to Exposures Scenarios. Scale of use, frequency of use and current or available engineering controls must be considered.

Definitions and abbreviations

PC—TWA: Permissible Concentration-Time Weighted Average
PC—STEL: Permissible Concentration-Short Term Exposure Limit
IARC: International Agency for Research on Cancer
ACGIH: American Conference of Governmental Industrial Hygienists
STEL: Short Term Exposure Limit
TEEL: Temporary Emergency Exposure Limit。
IDLH: Immediately Dangerous to Life or Health Concentrations
OSF: Odour Safety Factor
NOAEL :No Observed Adverse Effect Level
LOAEL: Lowest Observed Adverse Effect Level
TLV: Threshold Limit Value
LOD: Limit Of Detection
OTV: Odour Threshold Value
BCF: BioConcentration Factors
BEI: Biological Exposure Index

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end of SDS